

## Key Findings and Public Health Messages

- The California Department of Public Health (CDPH) received reports of the following cases with estimated symptom onset dates from 2009 through 2012:
  - 1,007 cases of *E. coli* O157 (average annual incidence rate of 0.67 cases per 100,000),
  - 674 cases of *E. coli* non-O157 (average annual incidence rate of 0.45 cases per 100,000), and
  - 165 cases of hemolytic uremic syndrome (HUS) (average annual incidence rate of 0.11 cases per 100,000).
- Reported incidence rates of *E. coli* non-O157 infection increased six-fold from 2009 through 2012 to essentially equal *E. coli* O157 infection rates by 2012 (0.79 and 0.76 per 100,000 rates of *E. coli* O157 and non-O157 infection, respectively).
- The average annual incidence rates for the four-year surveillance period were highest among:
  - *E. coli* O157 patients 1 to 4 years of age (3.47 per 100,000), 5 to 14 years of age (1.24 per 100,000), and children less than 1 year (0.89 per 100,000),
  - *E. coli* non-O157 patients 1 to 4 years of age (3.55 per 100,000) and children less than 1 year old (1.88 per 100,000), and
  - HUS patients 1 to 4 years of age (1.01 per 100,000), 5 to 14 years of age (0.23 per 100,000), and children less than 1 year (0.20 per 100,000).
- During the surveillance period, 108 (10.7 percent) *E. coli* O157 infections and 9 (1.3 percent) *E. coli* non-O157 infections progressed to HUS.
- From 2009 through 2012, there were 14 confirmed foodborne outbreaks of Shiga toxin-producing *E. coli* (STEC) involving 54 California case-patients. Thirteen (92.9 percent) of the outbreaks were confirmed to have been caused by *E. coli* O157, and 1 (7.1 percent) outbreak was caused by *E. coli* non-O157.
- Preventing contamination and cross-contamination during the processing and production of foods, avoiding raw and unpasteurized dairy products and juices, combined with consumer education may provide the best opportunities for preventing and controlling *E. coli* O157 and non-O157 infections and HUS.

## Background

Shiga toxin-producing *Escherichia coli* (STEC) are important enteric bacterial pathogens in the United States (US), causing an estimated 265,000 infections, more than 3600 hospitalizations, and 30 deaths each year<sup>1</sup>. These diarrhea-causing *E. coli* are named for the potent cytotoxins (Shiga toxins 1 and 2) they produce. *E. coli* O157 is the most frequently reported STEC serogroup in the US, causing approximately 95,000 infections, mostly by serotype *E. coli* O157:H7. The many other STEC serogroups, referred to in this report collectively as *E. coli* non-O157, cause approximately 170,000 infections nationwide each year<sup>2</sup>.

Exposure to the feces of a contaminated animal (STEC live in ruminant animals, like cattle) or an infected human can result in illness. Ingesting or handling contaminated food is a common cause of STEC, but illness can also result from direct contact with contaminated animals or their environments, consuming contaminated beverages, or direct exposure to infected people or their personal items<sup>2,3</sup>.

Acute illness, usually gastroenteritis, typically occurs after an incubation period of 3 to 4 days, but may occur anywhere from 1 to 10 days after exposure. Illness may be more severe in young children and elderly patients. Overall, *E. coli* O157 appears to be more likely to cause severe illness than *E. coli* non-O157, though illness severity is also affected by the virulence characteristics of the infecting strain<sup>2</sup>.

The national Healthy People (HP) 2020 target objective for *E. coli* O157 incidence is for an incidence rate lower than 0.60 cases per 100,000 population. There is no HP 2020 objective for *E. coli* non-O157 incidence.

About 5 to 10 percent of STEC case- patients develop hemolytic uremic syndrome (HUS), a delayed, life-threatening complication of a STEC infection. HUS is a disease characterized by hemolytic anemia, acute kidney failure, and often a low platelet count, and is the leading cause of short-term acute renal failure in US children<sup>4</sup>. Progression to HUS occurs on average 7 days after symptom onset, and may be delayed until after STEC infection has cleared<sup>2</sup>. Most cases the of HUS are caused by *E. coli* O157, but *E. coli* non-O157 can also cause HUS<sup>4,5</sup>.

For surveillance purposes, post-diarrheal HUS cases without laboratory evidence of an STEC infection are presumed to be related to an undetected STEC infection. The national HP 2020 target objective for

HUS incidence is for an incidence rate lower than 1 case per 100,000 children under 5 years of age. Described in this report is the epidemiology of *E. coli* O157 and *E. coli* non-O157 infections in California from January 1, 2009 through December 31, 2012 reported by December 4, 2014. The epidemiology of HUS is also described, including HUS cases in which STEC was identified and post-diarrheal HUS cases without laboratory evidence of an STEC infection. Data for 2012 are provisional and may differ from data in future publications. For a complete discussion of the definitions, methods, and limitations associated with this report, please refer to the Technical Notes<sup>6</sup>. The epidemiological description of STEC infections and HUS for the 2001-2008 surveillance period can be found in the Epidemiologic Summary of STEC-related infections and illnesses in California, 2001-2008<sup>7</sup>.

### **California reporting requirements and surveillance case definitions**

California Code of Regulations, Title 17, requires health care providers to report suspected cases of *E. coli* O157 infection, *E. coli* non-O157 (since late 2006) infection, and post-diarrheal HUS to their local health department immediately by telephone. Clinical and reference laboratories are also required to report laboratory testing results suggestive of *E. coli* O157 or *E. coli* non-O157 infection to either the California Reportable Disease Information Exchange (CalREDIE) (via electronic laboratory reporting) or the local health department; reporting must occur within one working day after the health care provider has been notified.

California regulations require local health officers to report to CDPH cases of *E. coli* O157 and *E. coli* non-O157 infection, and post-diarrheal HUS. California officially counted cases that satisfied the Centers for Disease Control and Prevention (CDC)/Council of State and Territorial

Epidemiologists' (CSTE) surveillance case definition of a confirmed or probable case<sup>8</sup>. During 2009 through 2012, the confirmed and probable case definitions for STEC infections were:

- A confirmed case was one with isolation of STEC from a clinical specimen. Serotype O157:H7 isolates were assumed to be Shiga toxin-producing, while for all other serotypes, evidence of toxin production or the presence of Shiga toxin genes was required.
- A probable case was one with isolation of *E. coli* O157 from a clinical specimen without confirmation of H antigen or Shiga toxin production, or a clinically compatible case that either was epidemiologically linked to a confirmed or probable case or had an elevated antibody titer to a known Shiga toxin-producing *E. coli* serotype. The confirmed and probable case definitions for HUS were:
  - A confirmed case was one with anemia with microangiopathic changes or renal injury evidenced by either hematuria, proteinuria, or elevated creatinine levels that began within three weeks of onset of acute or bloody diarrhea.
  - A probable case was one with laboratory evidence of HUS but an unclear history of diarrhea or a case that met all criteria for a confirmed case but did not have confirmed microangiopathic changes.

Cases of Shiga toxin detected in feces without further culture confirmation or serogroup identification are also reportable according to California regulation. This requirement was added in late 2006 because some commercial laboratories now test for Shiga toxin without subsequently confirming identification by culture or other means. However, Shiga toxin detected in feces without culture confirmation is not designated by CDC as nationally notifiable and lacks a standard CDC/ CSTE case definition, so is not described in this report.

## Epidemiology of STEC Infections and HUS

### *E. coli* O157 Infections

CDPH received reports of 1,007 cases of *E. coli* O157 infection with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.67 cases per 100,000 Californians. Since 2001, incidence rates fluctuated moderately: during 2001-2008, the rate ranged from 0.52 to 0.86 per 100,000, and during 2009-2012, the rate increased from 0.58 per 100,000 in 2010 to 0.79 per 100,000 in 2012 [Figure 1]. A total of 108 (10.7 percent) *E. coli* O157 infections progressed to HUS by the time of case report [Figure 2]. Of 299 *E. coli* O157 case-patients under 5 years of age, 55 (18.4 percent) developed HUS (not shown). During the surveillance period, two (0.2 percent) *E. coli* O157 case-patients were reported to have died by the time of case report.

During 2009-2012, the average annual incidence rates for *E. coli* O157 infection were highest among children 1 to 4 years of age (3.47 per 100,000), 5 to 14 years of age (1.24 per 100,000), and children less than 1 year old (0.89 per 100,000) [Figure 3]. The ratio of male to female case-patients was 0.9:1.0. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (18.7 percent). However, *E. coli* O157 cases with complete data reported White non-Hispanic race/ethnicity more frequently than would be expected based on the demographic profile of California [Figure 4].

County-specific average annual incidence rates of *E. coli* O157 infection during the surveillance period ranged from 0 to 7.86 per 100,000 [Figure 5]. Average annual incidence rates were 3.4 times higher in Northern California (1.12 per 100,000) than in Southern California (0.33 per 100,000). The Far North (1.85 per 100,000), San Joaquin Valley (1.23 per 100,000) and Central Coast (1.19 per 100,000) regions reported the highest average annual incidence rates during the surveillance period.

### *E. coli* Non-O157 Infections

CDPH received reports of 674 cases of *E. coli* non-O157 infection with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.45 cases per 100,000 Californians. Incidence rates for *E. coli* non-O157 infections increased by nearly 600 percent from 2009 (40 cases; 0.11 per 100,000) to 2012 (288 cases; 0.76 per 100,000). In contrast, rates were stable from 2006 (when reporting was first required in California) to the beginning of the current surveillance period (0.10 per 100,000 in 2006, 2007, and 2008) [Figure 1]. A total of 9 (1.3 percent) *E. coli* non-O157 infections progressed to HUS by the time the case was reported [Figure 2], and 2 (0.3 percent) case-patients were reported to have died.

The average annual incidence rates for *E. coli* non-O157 infection during the surveillance period were highest among children 1 to 4 years of age (3.55 per 100,000) and children less than 1 year old (1.88 per 100,000) [Figure 3]. The ratio of male to female case-patients was 0.9:1.0. Incidence rates by race/ethnicity were not calculated due to the substantial portion of missing data (13.2 percent). However, *E. coli* non-O157 cases with complete data reported Hispanic race/ethnicity more frequently than would be expected based on the demographic profile of California [Figure 4].

County-specific average annual incidence rates of *E. coli* non-O157 during the surveillance period ranged from 0 to 1.83 per 100,000. Average annual incidence rates were nearly the same in Northern California (0.46 per 100,000) and Southern California (0.44 per 100,000) [Figure 5]. The Central Coast (1.06 per 100,000) and San Diego (0.59 per 100,000) regions reported the highest *E. coli* non-O157 average annual incidence rates during the surveillance period.

### HUS

CDPH received reports of 165 patients with HUS with estimated symptom onset dates from 2009 through 2012. This corresponds to an average annual incidence rate of 0.11 cases per 100,000 Californians. Rates remained stable during 2009 to 2012 (range: 0.10 to 0.13 per 100,000), and have varied little since the 2001-2008 surveillance period (range: 0.09 to 0.14 per 100,000) [Figure 6]. The majority of HUS (108, 65.5 percent) diagnoses were associated with a laboratory-confirmed *E. coli* O157 infection, 9 (5.5 percent) HUS patients had an associated laboratory-confirmed *E. coli* non-O157 infection, and 48 (29.1 percent) HUS patients did not have laboratory evidence of an STEC infection [Figure 2]. Two (1.2 percent) HUS case-patients were reported to have died (one patient with a laboratory-confirmed *E. coli* O157 infection and one patient with a non-O157 infection, as described above).

Average annual HUS incidence rates were highest among children 1 to 4 years of age (1.01 per 100,000), 5 to 14 years of age (0.23 per 100,000), and less than 1 year (0.20 per 100,000) [Figure 3]. The ratio of male to female patients was 0.7:1.0. Incidence rates by race/ethnicity were not calculated due to missing data (15.2 percent). However, HUS patients reported White non-Hispanic race/ethnicity

more frequently than would be expected based on the demographic profile of California [Figure 4]. The average annual incidence rate for HUS for the surveillance period was 3.8 times higher in Northern California (0.19 per 100,000) than in Southern California (0.05 per 100,000) [Figure 5]. The San Joaquin Valley, (0.26 per 100,000), Far North (0.25 per 100,000) and Central Coast (0.19 per 100,000) regions reported the highest average annual incidence rates during the surveillance period.

### STEC Outbreaks

According to CDC's National Outbreak Reporting System data<sup>9</sup>, from 2009 through 2012, there were 14 foodborne outbreaks of STEC involving 54 California residents. Thirteen (92.9 percent) of the outbreaks were confirmed to have been caused by *E. coli* O157, and 1 (7.1 percent) outbreak was caused by *E. coli* non-O157. Of the 14 outbreaks, 10 (71.4 percent) involved cases exposed in multiple states (27 California residents were part of these multi-state outbreaks) and 4 (28.6 percent) outbreaks were confined to California (involving 27 case-patients). Among 13 (92.9 percent) outbreaks with a confirmed food vehicle, the most commonly implicated types of foods were beef (5, 38.5 percent) and vegetable row crops (4, 30.8 percent)<sup>10</sup>. The largest multi-state *E. coli* outbreak involving California residents occurred in 2009 and included 77 case-patients from more than ten states whose infection with *E. coli* O157:H7 was associated with consumption of cookie dough; 5 (6.5 percent) case-patients were CA residents. The largest outbreak confined to California occurred in 2012 and involved 12 cases of *E. coli* O157:H7 infection associated with romaine lettuce consumption. The lone *E. coli* non-O157 outbreak was a multi-state outbreak of *E. coli* O121 associated with consumption of a frozen meal; one California case-patient was involved.

Figure 1. California *E. coli* O157 and *E. coli* non-O157 infection case counts and incidence rates by estimated year of illness onset

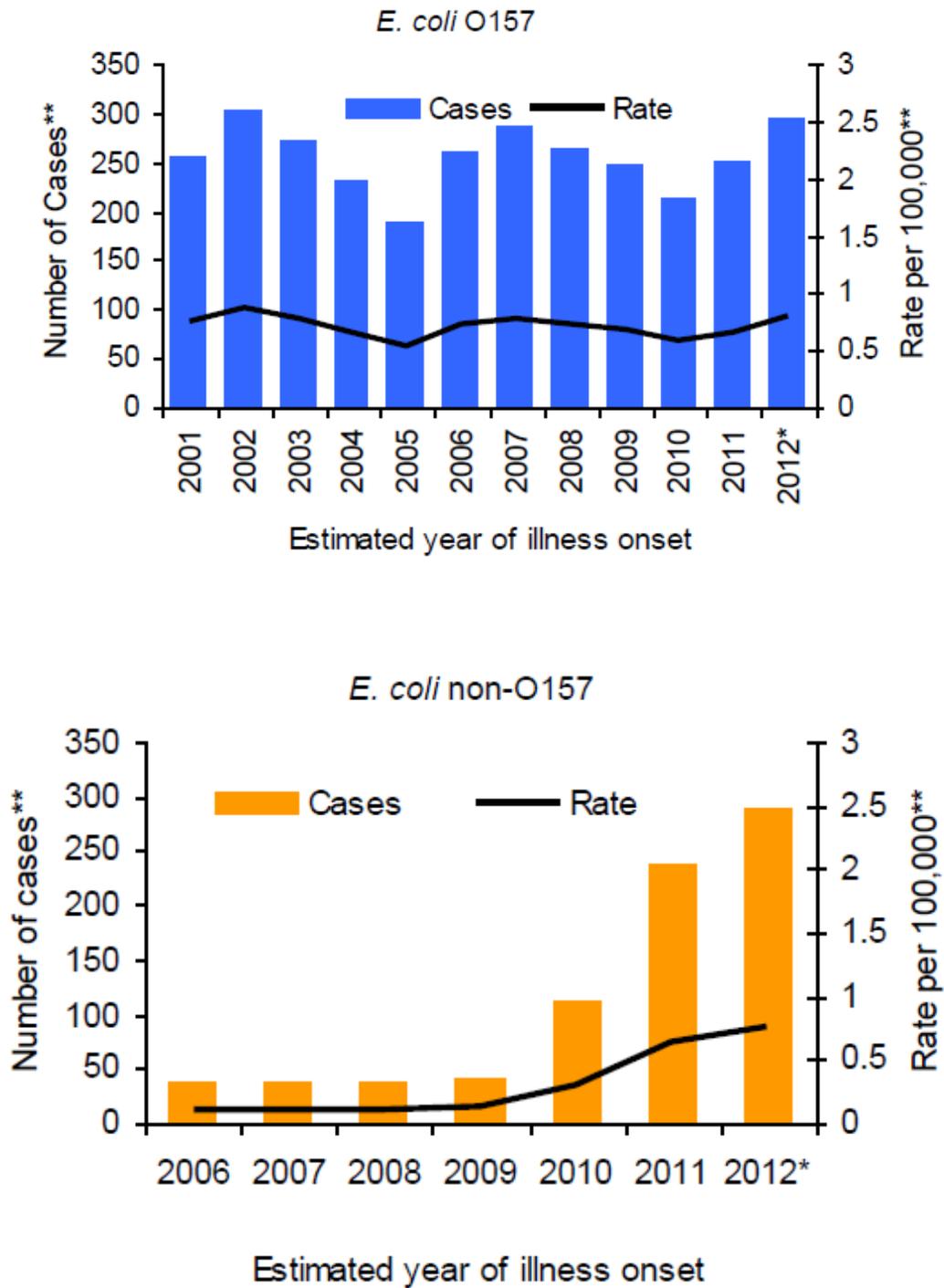


Figure 2. Venn diagram of California cases of *E. coli* O157 and *E. coli* non-O157 infection and Hemolytic Uremic Syndrome (HUS), 2009-2012\*

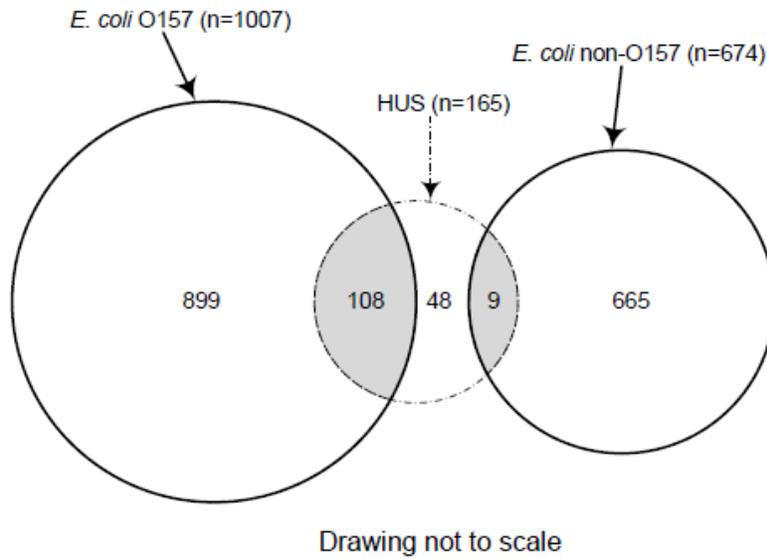


Figure 3. California *E. coli* O157 and *E. coli* non-O157 infection and HUS average annual incidence rates by age group, 2009-2012\*

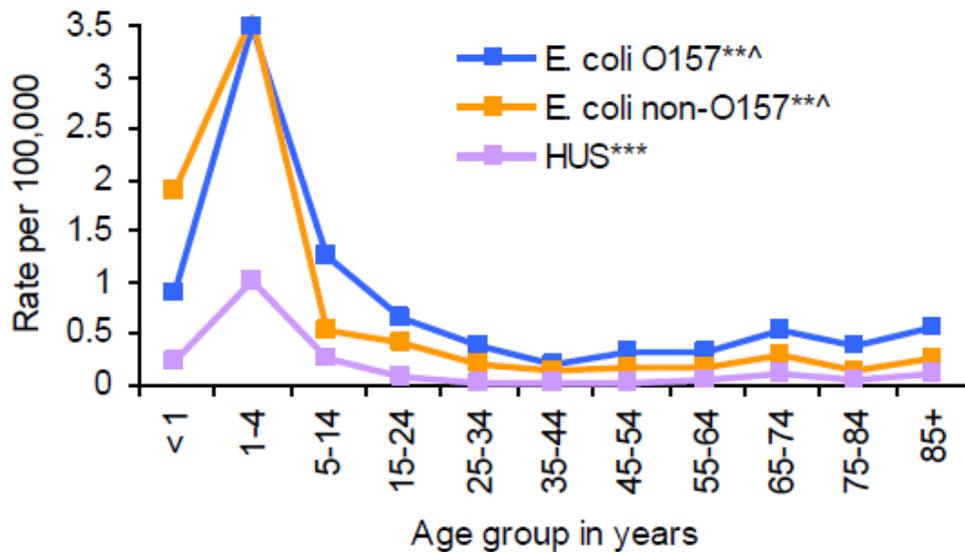
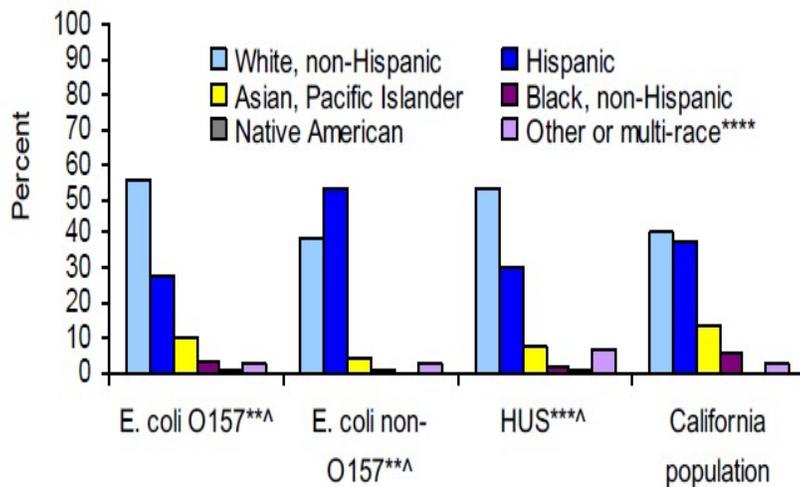


Figure 4. California *E. coli* O157 and *E. coli* non-O157 infection and HUS cases and population by race/ethnicity, 2009-2012\*



Notes for Figures 1-6

\*2012 data are provisional

\*\*Includes cases accompanied by HUS

\*\*\*Includes cases with laboratory evidence of STEC

\*\*\*\*Includes cases who identified 'other' as their race or more than one race and Californians ('population') who identified more than one race

^Unknowns were excluded

Figure 5. California county-specific *E. coli* O157 and *E. coli* non-O157 infection and HUS average annual incidence rates, 2009-2012\*

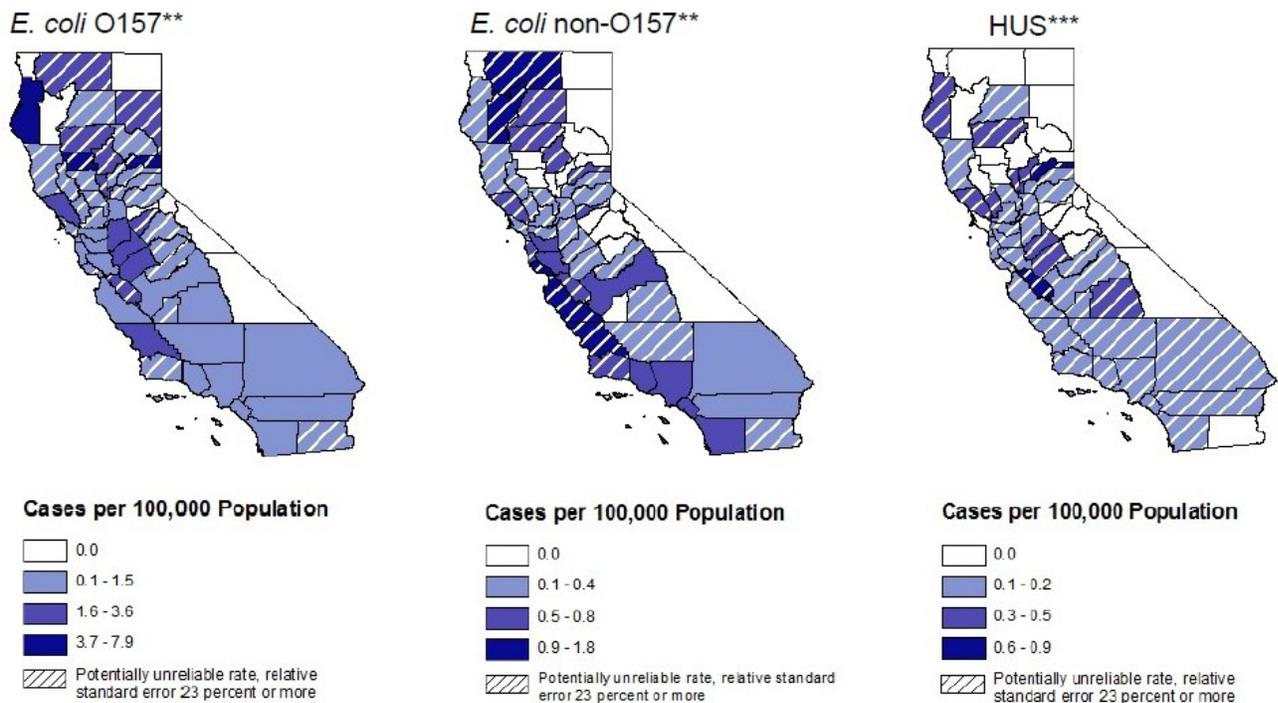
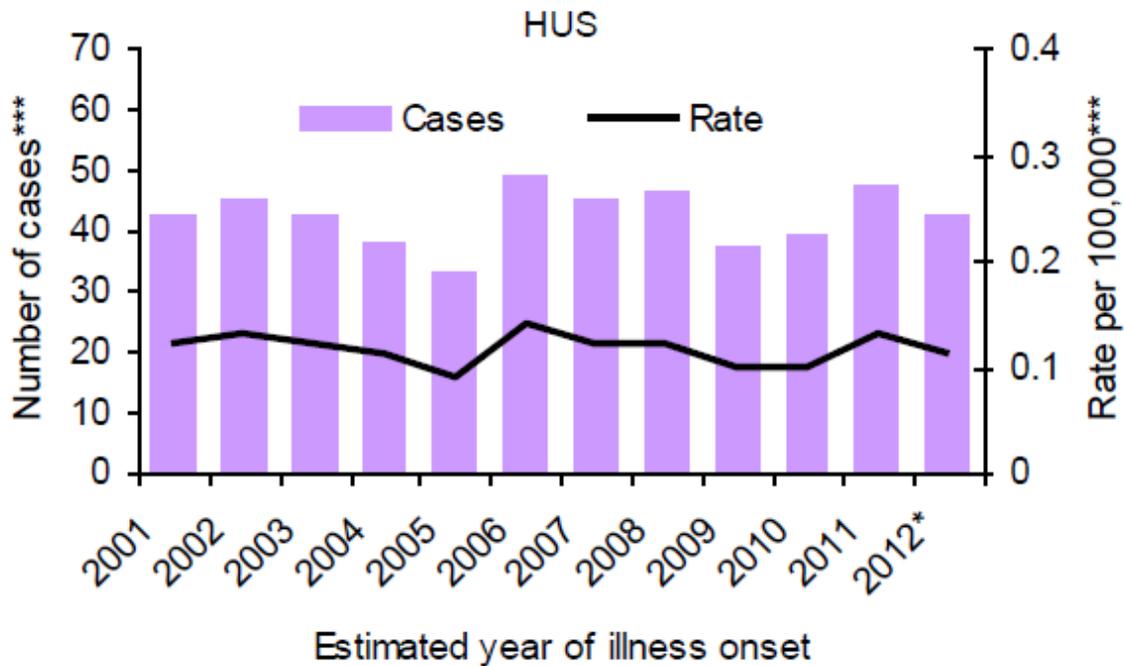


Figure 6. California HUS case counts and incidence rates by estimated year of illness onset



**Comment**

During 2009 through 2012, incidence rates of reported *E. coli* O157 infection among Californians fluctuated moderately but trended upwards towards the end of the surveillance period. The statewide average annual incidence rate of *E. coli* O157 infection for the surveillance period, 0.67 per 100,000, was just above the national Healthy People 2020 target objective of 0.60 per 100,000. *E. coli* O157 incidence rates among Californians during 2009-2012 were similar to those reported nationally<sup>11</sup>.

Incidence rates of *E. coli* non-O157 infection, which became reportable in California in 2006, increased six-fold from 2009 to 2012 to essentially equal *E. coli* O157 rates by the end of the surveillance period (0.79 and 0.76 per 100,000 of *E. coli* O157 and non-O157,

respectively, in 2012). This marked increase may be influenced by several factors, including increased use of Shiga toxin testing by clinical laboratories, growing awareness of reporting requirements for *E. coli* non-O157, and increasing numbers of Shiga toxin positive specimens forwarded to a public health laboratory for culture and identification. The rise in incidence may also be due to a true increase in *E. coli* non-O157 infections, due to yet undefined demographic and environmental risk factors. A rise in rates was also experienced in the US overall; *E. coli* non-O157 incidence rates, particularly in 2011 and 2012, were similar to those reported in the US<sup>11</sup>.

HUS incidence rates among Californians were relatively stable during 2009 through 2012 (average annual incidence rate of 0.11

cases per 100,000). Nearly 10 percent of all *E. coli* O157 and non-O157 infections reported during the surveillance period progressed to HUS by the time of their report.

However, as in the US overall, the majority of HUS diagnoses in California were associated with a laboratory-confirmed *E. coli* O157 infection, while only a small proportion were associated with a laboratory-confirmed *E. coli* non-O157 infection<sup>1,5</sup>.

Also similar to national trends, California children ages 1 to 4 years experienced the highest rates of *E. coli* O157 and *E. coli* non-O157 infection, as well as of HUS<sup>11,12</sup>. A slightly greater proportion (18.4 percent) of California *E. coli* O157 case-patients under 5 years of age had a HUS diagnosis than did nationally (about 15 percent)<sup>12</sup>.

However, the HUS annual average incidence rate in California children under 5 years (0.85 per 100,000) was below the HP 2020 target objective of 1 case per 100,000. During the surveillance period, the higher rate of *E. coli* O157 infections and HUS in Northern California compared to Southern California is notable. The reason for the difference in rates is unknown, and may reflect regional differences in demographics or exposures. Further investigation may be warranted.

The moderately fluctuating trend over time and annual average rate of both *E. coli* O157 infection and HUS among Californians during this surveillance period were similar to those of the last surveillance period. The age and race/ethnicity distribution of *E. coli* O157 and HUS cases during 2009-2012 remained fairly consistent with that of 2001-2008.

Rates of *E. coli* O157 and *E. coli* non-O157 infection may be underestimated for several reasons. Patients often do not seek medical attention or provide samples for diagnostic testing. Clinical specimens may not be tested properly, and isolates may not be forwarded to a public health laboratory for serotyping and strain typing. Laboratories and health care providers may not report STEC infections to local health departments.

Surveillance depends on the complete, timely, and accurate collection of data. In order to capture the burden of STEC infections in California and to develop a comprehensive public health response, it is crucial that clinical laboratories routinely test all stool specimens collected from patients with symptoms consistent with acute bacterial enteritis for the presence of Shiga toxin and attempt to culture STEC. Suspect STEC specimens must be sent to a public health laboratory for serogrouping and subtyping.

Preventing contamination and cross-contamination during the processing and production of foods, including beef and fresh fruits and vegetables, avoiding raw and unpasteurized dairy products and juices, combined with consumer education may provide the best opportunities for preventing and controlling *E. coli* O157 and non-O157 infections and HUS.

## References and resources

<sup>1</sup>[National Shiga Toxin-producing \*Escherichia coli\* \(STEC\) Surveillance Overview. Centers for Disease Control and Prevention.](http://www.cdc.gov/ncezid/dfwed/pdfs/national-stec-surveillance-overview-508c.pdf)  
<http://www.cdc.gov/ncezid/dfwed/pdfs/national-stec-surveillance-overview-508c.pdf>

<sup>2</sup>[\*E. coli\* \(\*Escherichia coli\*\). Centers for Disease Control and Prevention.](http://www.cdc.gov/ecoli/general/index.html)  
<http://www.cdc.gov/ecoli/general/index.html>

<sup>3</sup>[Escherichia coli O157:H7. California Department of Public](#)

[Health. http://www.cdph.ca.gov/HealthInfo/diseases/Pages/EscherichiacoliO157H7.aspx](http://www.cdph.ca.gov/HealthInfo/diseases/Pages/EscherichiacoliO157H7.aspx)

<sup>4</sup>[Hemolytic Uremic Syndrome in Children. National Kidney and Urologic Diseases Information Clearinghouse, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health. http://kidney.niddk.nih.gov/kudiseases/pubs/childkidneydiseases/hemolytic\\_uremic\\_syndrome/](http://kidney.niddk.nih.gov/kudiseases/pubs/childkidneydiseases/hemolytic_uremic_syndrome/)

<sup>5</sup>[Gould LH, Bopp C, Strockbine N et al. Recommendations for diagnosis of Shiga toxin-producing \*Escherichia coli\* infections by clinical laboratories. MMWR Recomm Rep. 2009 Oct 16;58\(RR-12\):1-14. http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5812a1.htm](http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5812a1.htm)

<sup>6</sup>[Epidemiologic Summaries of Selected General Communicable Diseases in California, 2009-2012: Technical Notes http://www.cdph.ca.gov/programs/sss/Documents/TechnicalNotes01-08and09-12.pdf](http://www.cdph.ca.gov/programs/sss/Documents/TechnicalNotes01-08and09-12.pdf)

<sup>7</sup>[Epidemiological Summaries of Selected General Communicable Diseases in California, 2001-2008: Shiga toxin-producing \*Escherichia coli\* \(STEC\). http://www.cdph.ca.gov/data/statistics/Pages/EpiSummariesCDsCA-01-08.aspx](http://www.cdph.ca.gov/data/statistics/Pages/EpiSummariesCDsCA-01-08.aspx)

<sup>8</sup>[National Notifiable Diseases Surveillance System, Case Definitions, Shiga Toxin-producing \*Escherichia coli\* \(STEC\) and Hemolytic Uremic Syndrome, Post-diarrheal. Centers for Disease Control and Prevention. http://wwwn.cdc.gov/NNDSS/script/casedef.aspx?CondYrID=665&DatePub=1/1/2006](http://wwwn.cdc.gov/NNDSS/script/casedef.aspx?CondYrID=665&DatePub=1/1/2006) 12:00:00 AM  
<http://wwwn.cdc.gov/NNDSS/script/casedef.aspx?CondYrID=699&DatePub=1/1/1996>

12:00:00 AM

<sup>9</sup>National Outbreak Reporting System (NORS), Centers for Disease Control and Prevention. Data extracted 11/12/2014.

<sup>10</sup>[Interagency Food Safety Analytics Collaboration \(IFSAC\). Centers for Disease Control and Prevention. http://www.cdc.gov/foodborneburden/attributions/partnerships.html](http://www.cdc.gov/foodborneburden/attributions/partnerships.html)

<sup>11</sup>[Shiga Toxin-producing \*Escherichia coli\* \(STEC\) National Surveillance Summary, 2012. Centers for Disease Control and Prevention. http://www.cdc.gov/ncezid/dfwed/PDFs/national-stec-surv-summ-2012-508c.pdf](http://www.cdc.gov/ncezid/dfwed/PDFs/national-stec-surv-summ-2012-508c.pdf)

<sup>12</sup>[E. coli Infection and Food Safety. Centers for Disease Control and Prevention. http://www.cdc.gov/Features/Ecolifinfection/](http://www.cdc.gov/Features/Ecolifinfection/)

Last updated 8/6/2015

Prepared by Kirsten Knutson, Farzaneh Tabnak, and Akiko Kimura, Infectious Diseases Branch

Acknowledging Jeff Higa for interpretation of outbreak data